----------oOo----------

THESIS

FOR THE DEGREE OF M.D.

BY

FRANCIS ALEX. HARDY, M.B.,C.M.

----------oOo----------

AN INQUIRY

INTO THE

ETIOLOGY, PATHOLOGY & CLINICAL FEATURES

OF

THORACIC ANEURYSMS.

----------oOo----------

I have taken up the question of Thoracic Aneurysm, partly because of its intrinsic interest & importance, and partly because I have had somewhat exceptional opportunities of studying the subject.

The differential diagnosis of Aneurysm from other morbid Thoracic conditions is sometimes excessively difficult, and requires the utmost skill and diligence, before one can come to a definite conclusion; but at the same time one cannot exaggerate its importance, for it is upon the accuracy of this diagnosis that the treatment must necessarily be based and the prognosis be determined.

My attempt here will be to discuss the subject in as concise a manner as possible, and to base my conclusions largely on my own observations in Hospital Wards, and in the Post Mortem room, modified to some extent by the opinions of recognised authorities in this and other countries. I may add that many of the cases hereafter referred to in the

tabulated lists have been obtained from the Clini-
-cal and Post Mortem records of the London Hospi-
-tals, while not a few described in more detail
have come under my own personal observation.

Galen defined Aneurysm in the following words:-
" Aneurysm is a dilatation or relaxation of a ve-
-nous vessel, with a dispersion of the spirituous mat-
-ter under the flesh, where it distributes itself
by jerks".

Aetius in the 6th century writes:- " Aneurysm
is a dilatation of vessels most frequently met with
in the throat, commonly happening to women in la-
-bour, on account of the forcible detention of the
spirits. The blood and spirits being poured forth
collect under the skin."

Fernelius in the 16th century approaches more
closely to the truth, and defines it as:- "the
dilatation of an artery full of spirituous blood."

For all practical purposes I would define aneu-
-rysm as a localised permanent dilatation of an ar-
-tery, which may be fusiform, or sacular according
to circumstances.

At the outset it might be as well to say some-
-thing regarding the etiology of Aneurysm, and in
the first place let us discuss its relation to the
pathological condition known as atheroma or

Endarteritis Deformans.

Atheroma which is a chronic inflammatory and degenerative change, occurring in the inner coats of arteries, especially the larger ones, is more or less a constant physiological process in old age, but it is not unfrequently observed in early mid-dle life, when there have been present such pre-disposing causes as Syphilis, gout, chronic alco-holism, chronic nephritis and lead poisoning, es-pecially when accompanied by hard manual labour. That there is a greater tendency for its occurence in certain families is a view strongly held by Dr. Strümpel, the Professor & Director of the Medical Clinique at Erlangen. That it occurs with greater frequency in men than in women is borne out by Post-Mortem statistics; certain vessels suffer more than others, and the aorta, the common site of Aneurysm, is probably the artery most liable to Atheroma. Certain arteries I may say very rarely suffer from this condition, and they are the Hepatic Artery, Gastric Artery and the Mesenteric Artery. Possibly if I may suggest a cause, because the collateral circu-lation prevents any abnormal rise in blood pres-sure. Further on, when I come to speak of Aneu-rysm in the lower animals, I shall point out that in horses the Anterior Mesenteric Artery is the commonest site of Aneurysm.

The question of increasing blood pressure and its relation to Atheroma I shall discuss further on. Atheroma when occurring in a large or middle sized vessel, usually starts as an Endarteritis passing outwards, causing Meaarteritis, and eventually a Periarteritis, but in a few cases the sequence of events seem to be in the reverse order, commencing as a Periarteritis and spreading inwards towards the Lumen. The changes of the Intima consist in a swelling of the Endothelial cells, and a prolifera-
-tion of the connective tissue cells immediately underneath, causing a roughening of this lining membrane. Fatty degeneration occurs in these patches, giving the charisteristic yellow translu-
-cent appearance. This may break down and form Atheromatous ulcers, or such patches may be scler-
-osed, and a deposition of limesalts occur on their surface, resulting in the formation of small cal-
-carious plates. Some similar changes occur in the Tunica Media and Tunica Adventitia which may also go on to fatty degeneration and calcification. The result of all this is, the vessels become rigid, elongated, tortuous and lose their elasticity. As a consequence of these changes, the expansile movement of the arteries is necessarily impaired, so that they cannot empty themselves, and a rise

in blood pressure must inevitably follow. This rise in blood pressure will make itself most felt at the part of the arterial system where the elas--ticity is weakest, and we shall get a greater or less dilatation.

Other degenerative changes of an arterie -scler--otic nature closely resembling those just described occur in the course of Tertiary Syphilis.

All authorities appear unanimous in holding the view that in the production of Aneurysm we must have arterie-sclerosis as a primary factor. Dr. Coale in his "Practical Essay on Aneurysm" * speaks of the uneveness and swelling of the inner tunic of the vessel, and the dirty brown appearance, and the brittle character of the middle tunic of the vessel in the early stage of Aneurysm formation. He quotes Rokitanski as saying " the affection of the artery plays the most important part in the produc--tion of Aneurysm, and this affection consists in excessive deposition occurring in the lining mem--brane of the vessel."

Dr.Guthrie, writing about this period looks upon the condition of Atheroma as purely of an in--flammatory nature, and Hodgson and Begin agree with him.

* Boston Medical & Surg. Journal 1862 vol LXV. p. 193.

Rokitanski on the other hand holds out that the process is entirely a degenerative one. Further on in his essay, he discusses the causes of Ather--oma, and mentions first Syphilis, then Mercurial Salivation, and lastly Alcoholism; but he speaks of them as views held by other men, and not as views of his own.

Dr. James Barr in his paper on the " Etiology of Aneurysm " * says " disease of the vessel wall is absolutely necessary", and then goes on to pro--pound the following theory:- "the Intima is nour--ished by imbibition, which must take place in great part during the period of the vessels repose, i.e. between the pulsations". Now when the blood pres--sure is high there is no true period of relaxation of the vessel, but a greater or less distension of the same, and this leads to a malnutrition of the Intima. The result is, a chronic inflammation occurs, which gives rise to a hyperplasia of the cell elements in the subendothelial layer of the Intima. This undergoes a retrograde metamorphosis in the form of a fatty degeneration, and the pro--duction of Atheroma. Allowing for the present that high blood pressure is a very important cause

*Liverpool Med Journal 1881-1882 vol I. p. 124.

in producing Aneurysm let us glance over the prin-
-ciple factors that give us a high blood pressure.
It may be brought about either by increase of resist-
-ance to the outflow through the capillaries, or
as is more common, by an increase in the force of
the inflow due to.excessive action of the heart.
We find this occurring in chronic Nephritis, severe
muscular labour, Alcoholism &c. When Atheroma is
once established, Dr. Barr believes any great de-
-mand upon one's physical powers, or any cause that
produces a sudden rise in blood pressure, is able
to produce an Aneurysm. He goes on to say:- Let
us consider a man to be overworked in some foundry
where physical work is great; what will happen is
this - 1. Hypertrophy or Hypertrophy with dilata-
-tion of the left ventricle will occur, and also
a slight increase of growth in size and capacity
of the right ventricle to meet this new demand.

2. Now follows a chronic inflammation and Ather-
-omatous changes in the Aorta and Aortic valves and
Coronary Arteries.

3. Following on the above, one of two things
must occur; a. the formation of Aneurysm and so a
reduction of the blood pressure, and the preserva-
-tion of the Aortic valves, or the reverse, namely:-

b. Valvular Lesion in the form of Aortic incom--petence, with first Hypertrophy of the left ven--tricle followed by dilatation, and then the usual history of back pressure through the pulmonary sys--tem, due to failing compensation.

If however we are to beleive this to be true, surely a large proportion of our cases of chronic Nephritis would have Aneurysm; for in them the blood pressure is notoriously high, yet Aneurysm is by no means common among them, in fact I should say rare, unless the case had some other history as Syphilis along with it. Further I would like to point out that surely in these cases there was Hy--pertrophy of the heart, and Dr. Barr admits this. Yet my personal experience has been, and I shall demonstrate this fact to you further on, that even the largest of Aneurysms of the Aorta are very often associated with no Hypertrophy, indeed I have seen very large Aneurysms with Atrophy of the heart. Dr. Barr, however, explains this in the following words:- " the tension falls from the patient not being able to keep it up with overwork and other causes, and the Aneurysm when large acts as a blood reservoir, and never at any time offers any great obstacle to the circulation; so that even if there be enlargement of the heart during the early stages

the compensatory Hypertrophy has time to disappear before you have an opportunity of examining the organs on the Post-Mortem table.

As to the exact cause of Aneurysm there is great variance of opinion. At the present day many are inclined to consider Syphilis as the only cause. I am quite willing to admit that Syphilis plays a very important part, and for this reason; it causes great weakness of the vessel wall, as say an Acute Aortitis , and what is equally important it does not confine the patient to the house, or make him unfit for work, and so with his vessels in this very unsatisfactory state, he goes on doing maybe severe muscular work. This induces a rise in blood pressure, which causes the artery to give way at the weak spot, and so an aneurysmal dilata-tion occurs at that spot. But I personally be-lieve that Syphilis is only one, though probably one of the most important, of the predisposing factors in the causation of Aneurysm; amongst which Rheumatism, gout and all conditions tending to a retrograde metamorphosis in the vessel walls must also be included; and that in all cases the exciting cause is a rise in blood pressure, which is usually produced by physical exercise or strain. This theory is borne out by the fact that Aneurysm

is most common among soldiers and men engaged in hard muscular labour (as witness our manufacturing towns, Newcastle, Birmingham &c.) than in any other class; and I believe this is why the treatment by Iodide of Potassium combined with Tufnell diet and absolute rest is so very popular with physicians, because it lowers the blood pressure to a minimum, and gives the blood vessels complete repose. Again prostitutes amongst whom Syphilis must be very common rarely suffer from Aneurysm. Such a class of women are not exposed to severe physical exercise, but lead an idle and lethargic existence.

As however Syphilis is now considered to be the important factor in the causation of Aneurysm, I shall give a fairly complete account of the latest views of eminent authorities on this subject. For this reason I would bring before you a most able research on this problem written by Dr. Franz Back--haus of Kiel University in the close of last year, under the heading " Uber Mesarteriitis Syphilitica und deren Beziehung zur Aneurysmenbildung der Aorta"* He says that during the last ten years much study has been devoted to the pathology of diseases of the Aorta. In 1885 Professor Doehle studied the matter and wrote on a case of his own occurring

* Beiträge zur pathologischen Anatomie und zur Algemeinen Pathologie von Dr Zeigler of Freiburg University Band xxii. Heft iii. 1897.

in a man. The Intima of the Aorta he says showed calcarious plates, but also showed other changes which were deeper in the walls of the vessel, deeper than the changes usually found in chronic endarter--itis. In such areas you could see distinct Aneu--rysmal bulgings; the microscope proved this and also showed that the changes were not like those of chronic Endarteritis; because strictly speaking in chronic Endarteritis the changes are only in the Intima, whereas in this case there were distinct changes going on in the Media, consisting of cell proliferation and granulation tissue formation; this seemed to him so typical of Syphilitic Arteritis that he placed it in that group. In 1895 Professor Doehle described two other cases that came under his notice, which showed exactly the same changes in the Media microscopically as in the latter case. He is convinced that all such cases must be regard--ed as Syphilitic, and he points out how very fa--vourable are these changes to the formation of Aneurysm.

Dr. Backhaus says that in discussing the Eti--ology of Aneurysm we must consider three methods:-

1. The Aortic walls may from some pernicious habits, or evil cause, or from great rise in blood

pressure, or from injury become torn in the muscu-
-lar coat or Elastic Lamina and become so weakened
that the vessel wall will bulge.

2. It may happen that the Media from some un-
-known cause be weakened and Aneurysm follow.

3. The arterial wall may from some inflammatory
process either in a localised area or over a diffused part
of the vessel become weakened in the elastic coat,
and the continuous blood stream makes it give and
bulge out.

To believe that every Aortic Aneurysm was due to
Syphilis would be wrong, but the majority certainly
are. Syphilitic inflammations must be regarded as
the commonest cause of all in cases of Aneurysm,
and those who have studied it bear this truth out.
The frequency of Aortic Aneurysm in Syphilitics is
a long recognized fact. In many cases we find
Syphilitic Mesarteritis and Atheroma occurring to-
-gether and then people are prone to regard the
latter and be blind to the former. Sometimes Ather-
-oma is present to so severe an extent as to almost
hide the presence of Syphilitic changes. This he
thinks explains why so many have gone wrong in
their views, as for example Professor Ziegler who
says that as soon as chronic Endarteritis has com-
-menced then things are in a favourable condition
for the production of Aneurysm. Dr. Backhaus does

not think this probable, but agrees with Professor Doehle that most are due to Syphilis; he discusses the work of Köster on this subject very freely and at much length and beleives him to be wrong in his views.

In 1888 Malmstein wrote a long article on the etiology of Aneurysm and which he here criticises. He considers Malmstein's research of great importance because they are the result of very accurate work at Post-Mortems, spreading over a graat number of cases, in all 101. Malmstein classified Aneu--rysms according to their etiology:-

Syphilitic	80%.
Senile	19%.
Traumatic, Erosion, Mycotic	1%.

Of these Syphilitic ones 90% occur between the ages of 26 and 50 years. The inner coat of the Aorta in these cases was most uneven and corrugated, often without any calcification or fatty degenera--tion, and only was there in a few cases inflamma-tory ulcers existing. The diagrams of his cases are so very identical in their likeness to those of Professor Doehle that one cannot help seeing he had the same clinical picture before him; the Intima was much thickened, uneven and corrugated. In the

Media there was no particular change around the Vasa-vasorum, but there was great cell proliferation here, the cells arranging themselves in rows.

Of the second variety, namely Aneurysms due to senile change there are 19%. and they occur usually about the age of 45 years. Microscopically Malm--stein found the following appearances:- Intima of Aorta was in no single case rough or uneven, but in some there was an ulcerated process going on; the Intima was thickened and there was present in it fatty degeneration and sometimes calcarious infil--tration; in the Media he found fatty degeneration of the muscular fibres, but no increase of the Vasa-vasorum; in the adventitia no change visible. This result is identical with the appearance of a case of chronic Endarteritis, and is quite different to the clinical picture of Syphilis which I showed you on the last page.

In 1895 Puppe wrote upon this subject in the " Deutsche Medic. Wochenschrift ". He examined 16 cases and says some of them microscopically were identical with the appearances described by Baum--garten as Syphilitic Arteritis. As regards ages:- three were over 60, and three between 50 and 60, and four between 40 and 50, one over and five under

36 years. cause of them:- Atheroma pure and simple in the two aged cases; Atheroma with Trau--matism in two cases; the remaining 12 were typical of Syphilis, seven of which acknowledged Syphilis during life. Microscopically these 12 cases demon--strated identically the same clinical picture that I have already placed before you, namely:- Much infiltration of the Vasa-vasorum in Media and Adven--titia, and around the Vasa-vasorum, and to some extent obliteration of the lumen of the vessel; much infiltration of cells and granulation tissue formation in the Media. The Elastic Lamina torn or ruptured in parts and the Intima much thickened. Professor Puppe, Doehle, Köster and some believe the Syphilitic inflammation starts primarily in the Media and Adventitia, and that the Intima suffers secondarily. Malmstein has studied this very accurately over a great number of cases and says that microscopically there is a very distinct Endar--teritis present in these cases and quite different to chronic Endarteritis, and he calls it a 'Gumma--tous-Sclerosis' because it is essentially Syphili--tic. Whether the changes commence first in the Media or in the Intima he does not say.

Summing up, Dr. Backhaus says that if we look at the works of Malmstein, Doehle, Koster and Puppe

and draw out our own conclusions we may say:- the disease of the Aorta in Syphilitics which produces Aneurysm is:- 1. Inflammatory. 2. It commences in the Media and the Adventitia participates in it. 3. It causes changes in the Intima in its later stages nothing to do with Atheroma. Up to within the last ten years all Aneurysms occurring in young people were associated with their work; Malmstein has found Syphilitic Mesarteritis in all such cases. Of Aneurysmsdue purely to Atheroma Malmstein has only seen one case and it was in an old man of 62 years of age, and therefore of the senile type.

All authorities do not seem so convinced about this; Dr. Barr says " I do not say that Syphilitic changes may not give rise to malnutrition and be associated with high blood pressure and so lead to the production of Aneurysm, but that it is a common factor I will not admit".

Dr.Timothy Holmes in delivering an address on this subject before the British Medical Society says " my own view is that the true Syphilitic de--generation of arteries rarely leads to Aneurysm, but rather to the obliteration of the small vessels which are its favourite site" *.

* British Med Journal 1886 vol II. p. 1147.

Dr.Barwell in criticising this paper supports Timo--thy Holmes regarding the relationship between Aneurysm and Syphilis. He says he has examined a very great number of Aneurysms, Post-Mortem, and also diseased arteries, and that he had not found in a single instance that the large vessels were affec--ted with Syphilitic manifestations comparable with the undoubted Syphilitic changes that were found in the smaller arteries.

That Syphilis affects the smaller vessels cau--sing a Syphilitic Endarteritis, Endarteritis Ob--literans and Thrombosis is admitted by all and is pointed out by Professor Greenfield, Drs. Gower, Buzzard, Sharkey in the "Transactions of the Patho--logical Society," Volume 28.

Professor Zeigler of Freiburg University says Aneurysm is usually the result of Arterio-scler--osis which weakens the vessel wall, impairing its elasticity and reducing its resisting powers. The larger Aneurysms are usually due to rupture of one or more coats and then also due to Arterio-sclero--sis. Sometimes however Aneurysm is due to simple Acute Arteritis.

In my notes on Post-Mortems that I have seen and on Museum specimens which I shall bring before

you shortly you will notice how very prominent a place Atheroma takes in the Statistics and how few cases show Acute Aortitis.

Chelius writes " Aneurysms arise either of their own accord or after determinate external influ--ences "; among the internal causes he gives us Rheumatism, Gout, Scrofula, Syphilis, Mercury and Chronic Alcoholism.

Reicherand noticed Aneurysms were most common among the surgical attendants of surgical amphi--theatres and attributed it to excessive drinking, and the carrying of heavy bodies into the theatre.

Dr. Guthrie is inclined to consider muscular exertion as the most predisposing cause,

Erichsen mentions climate, age. and cachexia, and remarks how much more frequent is the disease of Aneurysm in cold than in hot climates; he be--lieves that Syphilis may be a prediaposing cause of Aneurysm but he considers the statistics are not sufficiently great to warrant such a conclusion.

Professor Gross says * " mercury and Syphilis and the inordinate use of spirits are supposed to predispose to the formation of Aneurysm, but how

* System of Surgery. Edition vi. Vol. i. p 690—

far or in what degree remains to be demonstrated."

Bilroth writing on this subject says "special causes are little known; I am inclined to believe that the tendency to disease of the arteries as gout, is due to hereditary influences."

I have already mentioned that Erichsen in dis--cussing the etiology of Aneurysm mentions climate as a factor and that it is more common in cold than in hot countries, and in this Dr. Hayne of San Francisco supports him, in that he says * "the nature of the climate and the occupation of the people are two predisposing causes.

Dr. Sarazin likewise supports this idea by say--ing* that hot climates with great diurnal changes and humid atmosphere are very predisposing causes, and he produces statistics to support his views:- in Norway during the years 1875, 1876, 1877, there were 44,043 deaths and of these only four were due to Aneurysm; that is a rate per thousand 0.09

Now in Ontario during the year 1876 there were 18,623 deaths and of these 8 were due to Aneurysm representing a rate per thousand of 0.43.

In Michigan during the years 1870 to 1876 inclu--sive there were 85,189 deaths and of these 15 were

* Pacific Med Journal vol 7. p. 117.

due to Aneurysm, a rate per thousand 0.17.

As regards occupation influencing the produc--tion of Aneurysm we only require to look over our Hospital Records and think of our cases in private practice. My experience has been to find Aneurysm commonest amongst those who work in foundries and are exposed to the heat of the furnace and outside bad weather; a work associated with sweltering hard muscular movements accompanied with much thirst. I think this is why we find Aneurysm so prevalent in Newcastle and Birmingham; then again I have had many cases under me while I was Assisting House Surgeon and House Surgeon (protemps) in the Royal Infirmary at Southampton among dock labourers. I have also seen some remarkably good cases of Aneurysm occurring in butlers. In fact all occu--pations which combine hard muscular labour and thirst tend greatly I believe to multiply our cases of Aneurysm. Further on in my subject towards its close I have drawn up some statistics from the Brompton Hospital for Consumption and the Royal Hospital for Diseases of the Chest Records, and have dealt with all cases occurring between 1890 to 1896 inclusive, which demonstrate some of these points.

Professor Thoma in a long discourse upon the Etiology of Aneurysm * is very dogmatic in his views. He looks upon Atheroma as a compensatory process. He says "dilatation of the vessel occurs first, owing to some weakness in the middle coat of the vessel, and that part of the vessel is loaalised . The cause of this weakness in the Tunica Media he attributes to any cause that produces an abnormal rise in blood pressure, and which is persistent for a time. Following on this we get secondary ahanges in the Intima which he considers is a com--pensatory thickening, and he calls it Atheroma.

Professor Bäumler of Fr̀iburg University says † " as soon as Arterio-sclerosis has fully manifest--ed itself then everything is in a good way for the production of Aneurysm, and the explanation for this lies in the fact that the Arterial wall at that part has had its elasticity destroyed. He does not consider Syphilis to be an essential factor, though he admits that statistics point very strongly to it. He thinks that it may occur from Syphilitic Arteritis as in those of, the brain.

I have discussed the relationship of Syphilis to Aneurysm and have pointed out that it is probably the most important factor in its causation; but I also include under the same category all those

* Archives von Virchow upon Pathological Anatomy & Clinical Med. 1888 vol CX
† "Behandlung der Blutgefässkrankheiten" 1896.

conditions which help to bring about a retrograde matamorphosis in the body generally, and therefore in the tissues of the vessel wall such as the Acute Examthemata, the various toxaemias; be it the Syphilitic Virus or any other Virus, the various poisons acquired by habit and trade as Alcoholism, Chronic lead absorbtion, and also many of the other wasting diseases as malignant disease, Diabetis, and the many various disorders of the blood, as Leukaemia, &c. Many of these latter causes which I have mentioned as aiding the production of Aneu--rysm are not supported by much clinical evidence, but I attribute this lack of clinical support to the fact that in these conditions owing to the de--bilitated condition of the patient there is no accompanying rise in blood pressure.

All authorities are of opinion that Alcohol tends to promote an early degeneration of the ves--sels, and accordingly we should expect that Aneu--rysm would be fairly common among inebriates; the following letters however from different medical officers of such institutions show us conclusively that this is not so, and we are compelled to take the view that this fact is most probably due to the absence of severe manual labour, and physical strain amongst the inmates.

This fact further seems to demonstrate that Alcohol of itself alone is not sufficient to cause Aneurysm; the letters are as follows:- Dr. Blan-chard the Medical Superintendent of the Inebri-ate Home at Fort William writes " We have never had a death from Aneurysm in this Institution, nor any pronounced case of the same during the time I have been in charge, nor as far as I can ascertain previous to this".

Dr. Crothers Medical Superintendent of the Walnut Hill Lodge Inebriate Asylum, writes, " I have never met with a case of Aneurysm among the inebriates under my care; in the records of 700 cases which I have made, no cases of this kind have been noted."

Dr. Day of the Washington Home, Boston, writes " In the last 27 years I have treated a little over 10,000 Inebriates, and amongst all of these cases I have not in a single instance seen an Aneurysm of the Aorta."

Are we to, suppose for a moment that out of this great number of Chronic Alcoholics none of them had Atheroma or a fatty degeneration of their vessel walls? Surely it must be the very reverse with most of them. I attribute these surprising state-ments to, the following cause:- Just as much as the

aged man with a senile heart and diseased valves who sits in his arm-chair by the fire-side, leading a vegetative existence, exhibits so very few or even none of the symptoms of failing compensation, so these inebriates who lead a comfortable and pla--cid existence in these inebriate homes, with nothing to do, never have a sufficient rise of blood pres--sure to test the weakness of their vessel walls. Further the infrequency of Aneurysm in women al--ready alluded to, supports this theory.

Though most authorities are of opinion that Atheroma or Endarteritis is the all important pre--cursor of Aneurysm, and for my own part I may here mention, that I have made a careful study of all the Thoracic Aneurysms in the Royal College of Sur--geons, Museum, London, and have seen their speci--mens of very early starting Aneurysms, represented as mere dimples in the Aorta, and all of these were surrounded by patches of Atheroma, yet there are some authorities who contend that the primary changes occur in the middle coats of the vessel. Dr. Coates of Glasgow, in an address delivered be--fore the British Mediacl Association at Newcastle, in 1893, asserted that the causation of Aneurysm is usually an injury or wasting, or rupture of the Tunica Media, and in support of this urged that Atheroma is a disease of late life, while Aneurysm

is a disease of early middle life, when the blood pressure is highest. Recklinghausen, Eppinger, Manchot hold somewhat similar views.

I feel that my subject here would not be com--plete unless I drew your attention to what Virchow says;* he refers us back to the remarks of John Hunter and says " It is a fact which unfortunately has been forgotten, and now must be revived, namely that the strength of muscular contraction is a g great cause to consider, and that one gets a para--lysis of the Arterial coats in consequence, and the Elasticity is no longer sufficiently great to sup--port the vessel". He considers the Acute Exan--themata and all other diseases causing a Retrograde Metamorphosis in the body, and also those habits which would produce the same can weaken the vessel wall very considerably, rendering them fatty and brittle, and greatly impair their elasticity, so that a rise in blood pressure could cause an Aneu--rysm. In summing up his Article, he says:-

" Jede Entzundung, welche ~~eine~~ *eine* Zeitlang besteht, kann Erweiterungen, und Vergrosserungen der Gefasse des leidenes Theiles hervorbringen".

I have looked through one of the leading Veter--inary works of Medicine, and also one on Veterinary

* Archives of Pathology by Virchow 1895. vol iii.

Surgery, and I find that Syphilis is unknown amongst horses, or any of the lower animals. Experiments were done in Germany injecting Gonococci, and what were supposed to be Syphilitic Bacilli into rabbits and guinea-pigs with no result. Mr. Williams in his book on Veterinary Surgery, tells us that inflam--mation of the Arteries in the horse and in all the lower animals is a rare affection, and that when it does occur, it is usually a chronic Arteritis . he believes the cause of its occurence is generally due to injury. The Arteries most commonly affected are the Iliac Arteries, and these are imbedded in very powerful muscles which during contraction are liable to injure the Arteries.

Aneurysm is remarkably common in the horse; Dr. Brückmüller of Vienna examined 65 horses with a view to determine the frequency of Aneurysm, and found it was present in 59 of these, that is to say 91%; of these cases, 19 showed only a thickening of the Arterial coat, with or without slight dilata--tion of the vessel; 8 of the remainder were Aneu--rysms the size of a walnut; 29 were the size of a hen's egg. The age of these horses varied between 6 years and 20 years, though the majority were be--tween 6 and 11 years.Aneurysm is most common in the Anterior Mesenteric and the Posterior Aorta. The cause, he believes, is a degenerative change

in the Arterial Textures, or an ulceration in one or more of the coats. But here I would mention that in a great number of Aneurysms of the Anterior Mesenteric one found parasites in the sac wall namely "Strongylus Armatus" varietatis Minoris. Further this parasite is scarcely ever absent from the Mesenteric Artery of the ass, and this Artery is always thickened and calcareous, and very fre--quently you find Aneurysmal bulgings on it.

Here let me mention a very remarkable specimen I saw in the Royal College of Surgeons' Museum; it was a conical Aneurysm of the Aorta of a turtle, in size and shape like the drawing in the margin of page. The cause of its presence is difficult to explain, as the vessel looks quite healthy, and the sac wall likewise looks quite healthy, and there are no morbid manifestations to be seen anywhere.

Let us now pass on to the more important clini--cal features of Thoracic Aneurysm. The pressure symptoms are undoubtedly the most valuable in diag--nosing Aneurysm.. What these pressure symptoms may be, and how they may present themselves in the patient, I do not intend to enter into here in full detail, though their Clinical significance as regards the Diagnosis of the disease, and also in the local--isation of the site of the lesion cannot be over estimated. Some of the pressure symptoms however,

I would allude to here as they are not laid sufficient stress upon in the text-books.

Persistent and obstinate neuralgic pains which will not subside under any treatment, shooting pains down one arm which may be due to pressure directly, or indirectly on the Brachial Plexus, persistent intercostal neuralgic pains; all these should prompt us to examine carefully for Aneurysm.

Paralysis of one of the Vocal Cords and usually the left is a symptom which I believe to be of the utmost value. I have seen many cases of obscure Aneurysm, with almost no other symptoms except slight alteration of the voice, some Dyspnoea on exertion, and accentuated Aortic second sound; On examining the Larynx in these cases I have generally found a partial, if not complete paralysis of one Cord. The reason why more stress is not laid on this sypm--tom is I believe, because it is often over looked. Very often there is little or no change in the voice, certainly not enough to indicate a Laryngoscopic examination. But if the Laryngoscope were used in such cases as a matter of routine, one would be surprised to see how frequently we find partial paralysis present, the existence of which had been quite unsuspected. On first inspection there is little to notice about the Cords, except that the left one (the paralysed one suppose) appears a

little nearer the middle line, but if you ask the patient to take a long inspiration, then the condi--tion is obvious, for the Right Vocal Cord is abduc--ted nearly out of sight, the Left Vocal Cord re--maining stationary; now ask him to phonate, and in attempting a high note, the Left Vocal Cord does not move, but the Right Cord comes to the middle line, and attempts even to cross the middle line, to compensate for the paresis of its fellow; if there is on the other hand a complete paralysis of the Cord, then the condition is obvious at first sight, for the Left Cord assumes a 'Cadaveric Position', that is a position mid-way between phona--tion and ordinary breathing. I feel that I cannot lay too much stress on this symptom, for I have found it of the utmost value, not only in the Diag--nosing of Obscure cases, but also in the differen--tial Diagnosis between newgrowth and Intrathoracic Aneurysm. Except for the anatomical relationship between the arch of the Aorta and the left recurrent Laryngeal nerve; I cannot explain the reason why, but my experience has been that newgrowth in the Thorax much less frequently is accompanied by such paralysis.

Another symptom which I would draw your atten--tion to is inequality of the Pulses; sometimes we see cases of Aneurysm in which this is the only

symptom present, and though it is not in itself pathognomonic of Aneurysm, yet it is quite suffi--cient to make us consider the possibility of some Thoracic tumour being present, and so make us give a reserved Diagnosis. Let me here describe a case which I had through the kindness of Dr. R. Murray Leslie, the opportunity of examining in the Royal Hospital for Diseases of the Chest.

Edward Ayling; Photographer, 50 years of age; no family history; 4 years ago he had an attack of Haemoptysis, losing half a pint of blood; has had no recurrence of the same; 3 years ago he had in--fluenza; he has not had Syphilis.

Present illness; 3 years ago he had shortness of breath which has increased lately; 15 months ago he noticed a tumour to the right of the Sternum; 3 months after he felt pain in the tumour, followed by pain in left shoulder; about this time he devel--oped much huskiness, and then loss of voice; it is note worthy here to mention there was no cough de--veloped, and he never had one up to his death.

I found the patient lying on his back with an anxious expression, no great wasting, indeed during the last three or four weeks, he had been putting on a little weight since his admission into Hospital; no clubbing of fingers, face somewhat livid, skin clear and healthy, though of a somewhat cachectic

tinge, pupils were equal in size, reacting equally to light, conjunctivae glistening, pain and tender--ness in the right side of the neck, and right Axilla, but no glandular enlargement; in the left Axilla there was a large fairly hard gland, not freely movable, yet not distinctly tacked down; on removing the night-dress, I noticed the left chest distinctly bulged forwards in its upper half; the correspond--ing area on the right side seemed to be retracted, the intercostal spaces being evident here, but ob--literated on the left side. Over the right side and to the right of the Sternum is a swelling the size of a big orange, and occupying the 3rd, 4th, and 5th interspaces. Veins are prominent over the left side of chest and over this tumour on right side; there is deficient movement over the right side of chest.

On palpating the chest one found the Vocal Frem--itus greatly diminished over the upper four spaces of the left side; Cardiac impulse could not be detected on left side of chest; Radial pulses were equal; the tumour on the right side pulsated , but it is an Antero-posterior pulsation, not expansile.

The percussion note on the front and back of the right side is decidedly good except over the tumour where it is absolutely dull, and the patient flinch--es when you percuss over it. The percussion note

on the left side above the Clavicle and over the upper 3 spaces in front and behind is dull and wood--en, this dulness shades off obliquely into the Axilla; over the Cardiac Area there is an impaired note, but no true Cardiac dulness; in the Axilla the note is fairly good; posteriorly from Apex to base there is a general impairment of the note, dull at the Apex and impaired over the rest of the lung. There is a much greater extent of impaired breathing behind than in front.

On auscultating the right side of the chest the breath sounds were clear, no Aortic bruit; heart sounds very distinct over the tumour; but no bruits audible. On the left side there is an absence of breath sound all over; complete absence of breath sound over the upper 4 interspaces; diminution of and weak breath sounds audible over left lung posteriorly; Cardiac sounds are very distant, almost inaudible when listened for in their proper place. There was a well pronounced Accentuated Second to be heard over the tumour; the liver dulness is down to the Umbilicus; the Left Vocal Cord is paralysed; pulsation to the right of the Sternum became more marked towards death.

Patient suffered great pain; he was kept under morphia more or less all his latter days; the pain in the left shoulder was intense.

Post Mortem Report:- Speaking as an eye witness this is what I saw; heart was unduly small, all the valves healthy, and Aortic valves positively competent, it was situated on the right side under the 3rd, 4th, and 5th interspaces, and between it and the chest wall htere was an Aneurysm of the ascending limb about the size of an orange; the Aneurysm was limited above at the distal margin of the Innominate Artery by a firm hard cartilaginous ring about the size of a half crown; the Aneurysm commenced immediately above the valves inside the Pericardial sac; Innominate Artery was not involved. The Aneurysm as I said was limited by a ring above, and through this you came into another Aneurysmal sac, one of the largest I have ever seen. It ex--tended from the distal side of the Innominate Ar--tery down to one inch above where the Aorta passes through the Diaphragm, i.e. it involved the Trans--verse Arch, the descending limb, and the descending Thoracic Aorta; it was a sacular Aneurysm like the other, but much larger; it occupied nearly the entire left chest, the lung having collapsed. This enormous sac was lined with laminated blood clot to about 3 inches thick, and the channel through which the blood burrowed its way was full of Post Mortem clot; the sac had ruptured at the lower part anteriorly of this large sac and gone into the left

Pleural cavity.

I have endeavoured to describe the case briefly and yet to bring in all its clinical features, to demonstrate the difficulty in making a diagnosis, and to point out some of what appeared to me as the chief diagnostic symptoms. Here we had paralysis of the Left Vocal Cord well marked on examination though his voice was only a little thick when talk-ing. Further he had a well marked Accentuated Second sound heard over the tumour on the right side. Further since his admission he had been actually gaining weight, not very much but still a recognisa-ble quantity every week; there were no manifesta-tions of Syphilis during life or at the Post Mortem.

I might add that many of the members of the staff after repeated examinations came to the con-clusion that the case was probably one of new growth.

Returning to where I left off in discussing the pressure symptoms of Aneurysm, I would mention here how I have noticed in isolated cases that patients have never complained of pain in the back, and yet at the Post Mortem, one has found very extensive erosion of the Vertebral column. This absence of pain under such circumstances only happens occasion-ally, but that it should occur is sufficient to make one always be on one's guard.

Some text books dwell much upon the brassy Aneu--rysmal cough as a cardinal symptom; I am willing to admit that its presence is strongly indicative of an Aneurysm, but my experience has been that it is not unfrequently absent even when on a Laryngoscopic examination there is quite an appreciable amount of Vocal Cord paralysis present.

A slightly blood stained expectoration ia a symp--tom which must never be neglected. This is not unfrequently accompanied by a diminution of breath sounds over one or other lung; these two symptoms are occasionally the only indications pointing to the existence of a deep seated Thoracic Aneurysm. Of course the blood stained sputum may have another significance and may serve to warn us of impending rupture into the air passages.

Perhaps I should say something here about Dysp--noea as a pressure symptom, as it causes such great distress to the patient. The cause of it I attri--bute to pressure on the Trachea, or on the left or right Bronchus, or it may occasionally be induced spasmodically by pressure on the Pneumogastric nerves. When I was in the Wards of the Royal Hospital for diseases of the Chest one afternoon, I saw a very typi--cal attack of Dyspnoea come on in a case that proved at the Post Mortem to be Innominate Aneurysm and Fusiform Dilatation of the Arch. He was sitting

quietly in a chair when he was suddenly seized with great Dyspnoea, the breathing was very laboured in--deed, and accompanied with a very loud Stridor which could be heard all over the Ward; the face was florid and very dusky, the ears, lips, hands, and nails very cyanosed; the pulse was of very high tension; he was given Nitrite of Amyl on cotton wool, and in some five to ten minutes it had a very good effect; the pulse becoming much softer and the Dyspnoea with its unpleasant sequelae passed off. A subcutaneous injection of morphia however in my experience is the best remedy to employ in such cases.

In the majority of cases Aneurysms occur on the right side of the Mid-sternal line and along with it there we find its accompanying physical signs; This I attribute to a fact which I have already pointed out to you in my Statistics, that Aneurysm of the ascending limb is by far the commonest vari--ety, and as Dr. Oswald Browne has shown* nearly all of these grow from the Dextro Anterior aspect of the Arch, and grow therefore towards the right and upwards. He also points out that those few cases which develop from the concave aspect of the Arch,

* 'Aneurysms of the Aorta.' 1895.

invariably grow to the Left side of the Chest. That the Aneurysmal swelling may be present on either side of Chest, or between the Left Scapula and Spine behind is understood, for one sees occasional illus--trations of these varieties too in our Hospitals.

In Diagnosing between an Aneurysm and a tumour lying immediately over a vessel through which pulsa--tions are conducted, Dr. Richardson* lays much stress upon the value of Sphygmographic tracings. If we take a tracing of the Radial Pulse first and then a tracing over the tumour, if this tumour be an Aneurysm, we shall get the same pulse wave as at the wrist in its outline, though it may be bigger or smaller depending upon respiratory movements. If however the tumour is a solid body with Arterial Pulse conducted through, then we shall get no pulse wave, but only an up and down stroke. Further if we get a pulse wave we must keep in mind a condition which I shall speak about soon, and was pointed out by Dr. George Balfour, i.e. the exposed Pulmonary Artery, and exposed Aorta. In the text books we are told such swellings pulsate, and that the pulsa--tion is an expansile one; this is quite true, but we must bear in mind the fact that some Aneurysmal

* Asclepaid vol. I. p 354.

tumours do not pulsate at all. In the case I described of the man Edward Ayling, the larger of the two Aneurysms did not pulsate at all, though it was bulging forward the left chest. Mr. Baker, late surgeon of St. Bartholomew's Hospital* discusses this and relates several cases, where there was an Aneurysmal swelling and no pulsation whatsoever. These cases were the cause of much discussion, and diversity of opinion as to their nature.

But supposing we have a very palpatable swelling which produces a Sphygmographic tracing like the one at the wrist, and over it we can hear a Systolic bruit and an Accentuated Second, must we necessarily call that Aneurysm ! Dr George Balfour of Edinburgh was the first to draw attention to this†; he de--scribes 3 cases of pulsating tumour on the left side of the chest, accompanyed with Accentuated Second sound; in each case it was in the second left interspace; percussion note was very dull over the Area, and a bruit Systolic in time could be heard in each case. At the Post Mortems no Aneu--rysms were to he found, but in each case the left lung was retracted upwards and backwards, from dis--ease, leaving bare the Pulmonary Artery, which allowed of it coming close up under the Chest wall.

* Hospital Reports of St Bartholomews 1879. Vol xv. p75-84.

† Edinburs Medical Journal. 1871. vol xvi. p.702-715.

Hence we get a pulsating tumour in the Second interspace The phenomina he explains as follows; the apparent--ly accentuated second sound is really only the closing of the pulmonary valves, but heard more loudly than usual because the pulmonary Artery is bared, and allowed to come to the surface; the Sys--tolic murmur is due to the pulmonary Artery no longer having the support of the surrounding lung substance, and so the Ventricular contraction causes kinking of the vessel, pressing it against the chest wall.

Sir Douglas Powell* described cases where the Aorta was the exposed vessel, and not the pulmonary Artery ; he then goes on to discuss vaso-motor-paresis of the Aorta as giving rise to a palpatable thrill and a murmur.

The difficulties which such cases cause when we are desirous of making a differential Diagnosis are very considerable. Dr. George Balfour would have us bear in mind that usually the pulsation over an Aneurysm is stronger than the Apex beat, and the pulsation over an exposed vessel is weaker than the Apex beat.

As regards the use of percussion as an aid to the Diagnosis of Aneurysm, I think I may dismiss it

* British Medical Journal. 1889. Vol II p 1336+1394.

with very few words; its assistance is of the most value in that it is accurate in mapping out areas of dulness, be they areas occupied by solids or by fluids; as an aid also to localising the position of an Aneurysm -or tumour, it is very serviciable. Further the dulness caused by Aneurysm is usually more or less rounded in outline, while in the case of Thoracic newgrowth it is much more frequently irregular.

The value of auscultation to us is very great in the Diagnosis of Aneurysm, and if I may say so it is in this disease of the Circulatory system that the Stethoscope is of such value. In Aneurys--mal swellings we usually hear a Systolic murmur over the sac and an Accentuated Second over the heart; these are very valuable signs to us and almost pathognomonic, and when taken with other symptoms the Diagnosis of Aneurysm is practically certain. We must of course bear in mind those ex--ceptions I have mentioned as described by Dr. George Balfour. This murmur however, may be entirely ab--sent even over a large pulsating Aneurysm (vide case described) but there is usually present an Accentuated Second sound, the importance of which cannot in my mind be over estimated. In my

experience its absence is almost sufficient to nega-
-tive the presence of Aneurysm.

Sir Douglas Powell, in writing on the Diagnosis of Aneurysm* says he considers a Dyastolic murmur of great Diagnostic value for it furnishes evidence of intra-arterial disease, and if associated with pressure sounds of tumour then the Aneurysmal nature of that tumour is almost demonstrated. He also lays much emphasis upon the presence of a Systolic murmur combined with an Accentuated Second sound.

Dr. Fenwick and Dr. Overend have raised the question of the value of Intra-thoracic auscultation and Intra-thoracic sphygmographic tracings being taken in cases of suspected Aneurysm.† The method consists in passing a soft oesophegeal tube down the oesophagus, which has an elastic membrane capable of being inflated; the external end is in contact with a Marey's Tambour, and in connection with a revolving cylinder. When the distal end of the tube is over the Aneurysm or tumour then tracings are taken; by removing the Marey's Tambour we can attach a stethoscope and auscultate it. I myself have had no experience with this, but if I was at all in doubt, and it is , I suppose

*. British Medical Journal 1889. Vol II p1336-1394.
†. British Medical Journal 1893. Vol I. p.286.

only in obscure cases this method would be adopted, I should hesitate in the interest of my patient to make such an attempt, as it would in all probability precipitate the end by causing early rupture.

I have taken the trouble of carefully examining the Clinical and Post Mortem Records of nearly a hundred successive cases admitted into some of the London Hospitals during the last few years, and I must here express my thanks to the Physicians and Pathologists for so kindly permitting me to have access to their case-books; my thanks are specially due to Dr. Habershom and Dr. R. Murray -Leslie for showing me some of their cases.

As will be seen from the accessory tables, I have classified the more important points under their appropriate headings. From these tables I have drawn the following conclusions:- Aneurysm is vastly more common amongst men than amongst women, and for reasons that I have already mentioned; when occur--ring in females it seems to show itself at the same period in life i.e. about the age of 45 years though there are exceptions; also women the subjects of Aneurysm are usually laundry women or charwomen or are otherwise engaged in hard physical work.

Emaciation is the exception and not the rule in subjects with Aneurysm; the cases in Hospital invariably gain weight even up to the date of their death.

As regards age my statistics show the mean age amongst both men and women to be 45 years.

That Aneurysm most commonly affects the ascend--ing limb of the Aorta is very strongly borne out by all statistics. Dr. Oswald Browne* has pointed out that the majority of these grow from the Dex--tro anterior aspect of the Aortic arch and grow to the right, and very few grow from the left wall of the ascending limb; but when they do do so they grow to the left side of the chest; the proportion of Aneurysms growing to the left is exceedingly few. In my own statistics I can only show two cases, all the others grew to the right and arose from the Dextro anterior aspect of the wall.

The heart in the majority of cases is certainly not Hypertrophied, and when it is so we must bear in mind the advanced general Atheroma of the entire systemic system; also we must remember Aneurysm often causes very much displacement of the heart, and may so deceive us.

The Innominate Artery is rarely affected with Aneurysm, the mean average I place at 7.7%

The pulmonary Artery in its first part is ex--ceedingly rarely the site of Aneurysm; I have described one solitary case amongst my statistics and Dr. Oswald Browne who has classified 631 cases

* "Aneurysms of the Aorta." 1895.

occurring in St. Bartholomew's Hospital, could not find a single case.

ANALYSIS
of the
TABLES OF STATISTICS.

1st part was alone affected in.....	24·1%
1st & 2nd parts together affected in...	19·9%
2nd part alone affected in.....	18·7%
2nd & 3rd parts together affected in...	14·3%
3rd part alone affected in.....	5·5%
Entire arch was affected in.....	6·6%
1st & 3rd parts were affected in.....	1·1%
Descending Thoracic Aorta affect. in...	2·2%
Innominate Artery affected in	7·7%
Pulmonary Artery affected in.....	1·1%

The proportion of men to women the subjects of Aneurysm is as 84·6 : 15·4

As regards age, men and women seem to be affected at the same period in life, and I calculate it to be 45 years.

In examining the heart I found in every case an Accentuated Second sound in the Aortic area, and very frequently a bruit over the base of the heart, usually Systolic in time, sometimes Dyastolic and occasionally both. In referring to the question

of Hypertrophy of the heart in these subjects, I have made my Analysis from the Post Mortem Records, as I consider the fallacies that may arise in Clinical Records to be considerable, namely displaced heart and sometimes the Cardiac dulness is obscured, from pulmonary and other diseases. My statistics show that Hypertrophy of the heart occurs in 66·7%

The larynx I found to be affected in 46·2% of the cases examined; the left cord was the one para--lysed in all the cases except three, and the part of Aorta involved in these cases always included the transverse part of the arch.

My statistics show that Syphilis was the cause of Aneurysm in 39·8% of the cases.

I have drawn up an Analysis of the symptoms occurring in the cases that form my statistics, and have arranged them so that they compare altogether side by side; this Analysis will be found at the close of the tables, at the end of the paper. I have carefully avoided analysing the cases of Hae--moptysis, because many of these patients were phthisical, neither have I analysed the cases show--ing symptoms of Dyspnoea, because I found this symptom to be invariably present in all cases to a greater or less degree.

BROMPTON HOSPITAL FOR CONSUMPTION:-

----------oOo----------

CLINICAL RECORDS 1890 - 1896 (inclusive).

----------oOo----------

Sex.	Age.	Part of Aorta.	State of the Heart.	Effects of the Aneurysm.	Larynx.	Cause.	Result.	Observer.
M.	38	2nd.part.	Apex beat 5th space I.N.L. some dilatation. No bruit, Second soun accentuated.	Visible pulsation & bruit Second l.interspace; venous injection here impaired note; pressure on S.V.C. dyspnoea on exertion.	L Vocal cord has impaired movement.	Syphilis	Improved	Dr.Tatham
M.	35	1st part.	Apex beat in 5th space I.N.L. No bruit.	Dyspnoea on exertion; impaired note & Tracheal breathing over 2nd & 3rd right cartilages.	No note.	Been a soldier. ? Syphilis	Improved	Dr. Reginald Thompson
M.	26	Dilated arch	Normal. Accentuated Second.	Haemoptysis & Dyspnoea on exertion	No note	Sailor No Syph.	Improved	,,
M.	47	1st part	Systolic bruit all over and Accen--tuated Second; sometimes a dyas--tolic bruit.	Dulness of Manubrium, Systolic Bruit all over & Accentuated Sec.		No note.	Improved.	,,
M.	35	1st part	Apex beat not felt; sounds faint; Cardiac dulness obscured.	Dulness over 1st & 2nd R. Inter--spaces. Haemoptysis. R.Radial Pulse > L.Radial Pulse.	Normal	None.	Improved.	,,
F.	46	1st part	Systolic bruit at Apex; booming & accentuated second in Aortic area	Pressure on Trachea, R.R.Pulse > L.R.Pulse, Dulness from R.Clavicle to 3rd rib, no breath sounds, Sys--tolic Bruit, Accentuated Second.	Normal.	No Syph.	Relieved.	,,
M.	45	2nd part	Apex beat in 5th space I.N.L. hear--ing of Precordia; First Booming Musical Bruit, diastolic in time.	Pulsation in R.Supra & Infra Clavic fossae. Carotid L.Carotid.	Normal.	No Syph.	Improved.	,,
M.	34 ?	Aneurysm.	Normal	Slight Haemoptysis, Some Dysphagia & other vague symptoms.	Normal.	No Syph.	Improved.	,,

BROMPTON HOSPITAL FOR CONSUMPTION:-

----------oOo----------

CLINICAL RECORDS 1890 - 1896 (inclusive).

----------oOo----------

BROMPTON HOSPITAL FOR CONSUMPTION.

----------oOo----------

CLINICAL RECORDS 1890 - 1896 (inclusive).

----------oOo----------

Sex	Age	Part of Aorta	State of the Heart.	Effects of the Aneurysm.	Larynx	Cause	Result	Observer.
M.	20	2nd & 3rd.	Normal	Haemoptysis, Dyspnoea, Cough Dulness in Supra & infra Clavic regions; Sys-tolic Bruit here, no Pulsation, R.R. Pulse > L.R.Pulse at the wrist.	Normal	No note	Improved	Dr Thomp-son.
M.	48	Innominate Artery.	A.R.in 5th space I.N.I., heaving, loud systolic over Xyphisternum, Sec. sound Accentuated.	Pulsation in neck on both sides Dulness over R.Clavicle & down right side of Sternum; Tracheal breathing marked Dyspnoea, Marked pulsation of Sternal end of Clavicle.	No note.	Syphilis	I.S.Q.	,,
M.	47	ist & 2nd	A.B.X.N.L. Systolic Bruit at the base, Accentuated Second.	Great Dyspnoea & Brassy Cough, Dul--ness over Manubrium downwards & out--wards for one inch on either side some pulsation, Alteration in pulse.	No note	Rheumatic fever	Improved	,,
M.	41	1st part	Hypertrophied, Accentuated Second.	Dulness to right of Sternum.& 2nd & 3rd interspaces.	No note	No Syph	Improved	,,
M.	41	1st part	Second sound Accentuated.	Dulness to right & left of Sternum & over Manubrium.	,,	No Syph	Improved	,,
F.	28	2nd part	A.B.in 5th space I.N.I. Accentuate Second, Booming First, No Bruit.	Stenosis of left Bronchus, Cough, Dyspnoea, Dilated veins on Chest, ex--ternal jugulars prominent.	L.V.cord Paraly--sed.	Syphilis	Improved	,,
M.	49	1st part	Heaving diffuse impulse, Systolic Bruit at Apex, Accentuated Second	Visible Tumour over Manubrium, Pulsa--tion very markedly seen & felt, No thrill, Systolic Bruit.	No note	No Syph	Post Mor--tem I. 139	
M.	44	1st part	A.B.in 5th space I.N.L. Double Bruit at the base	Dulness right of Sternum, extreme Dyspnoea.	No note	No Syph.	Post Mor --tem	,,

BROMPTON HOSPITAL FOR CONSUMPTION.

----------oOo----------

CLINICAL RECORDS 1890 - 1896 (inclusive).

----------oOo----------

BROMPTON HOSPITAL FOR CONSUMPTION.

----------o0o----------

CLINICAL RECORDS 1890 - 1896 (inclusive).

----------o0o----------

SEX	AGE	PART OF AORTA	STATE OF HEART.	EFFECTS OF THE ANEURYSM	LARYNX	CAUSE	RESULT	OBSERVER.
								Dr Acland.
M.	43	First Part	Hypertrophied & Double Bruit at the base.	Visible pulsation in vessels of neck, no pulsation elsewhere,	No note	Syphilis Alcohol	Post	
M.	48	1st & 2nd & 3rd	A.B.in 5th space I.N.L. Booming First, Accentuated Sec. No Bruit,	Pulsating tumour right of Sternum left jujular distended Brassy Cough Pulsation in Episternal notch, Sys--tolic Bruit over tumour, Tumour very visible.	Left V.cord paraly--sed	No Syph.	Post Mortem F.165.	,,
M.	45	2nd & 3rd	Normal size Hystolic Bruit at base Accentuated Sec.	Dilated R.Pupil, left pulse > R.Pulse, Dulness to left of Manubrium, Haemoptisis, Systolic Bruit over the Tumour, Dyspnoea & Angina pains	R.V. Cord impaired movement	Syphilis	Improved	,,
M.	31	1st part	A.B.in 5th space I.N.L. Systolic Bruit & Accentuated Sec.	Visible Tumour right of Sternum pulsating , Systolic Bruit, husky voice.	Cords not seen	Syphilis Alcohol	Improved	Dr Green
F.	46	? Part affected	Enlarged Systolic Bruit, Accentuat Second.	Much Dyspnoea, occasional Aphonia Functional Paresis of Cords	Normal	None	Improved	,,
M.	46	1st & 2nd & 3rd	Cardiac dulness lost in emphysema, Sounds lost also.	Visible Tumour in upper ½ Sternum Dull note from Episternal notch to third rib, also for 1 inch on either side, pulsates, Bronchial breathing, Respiration sometimes stridulous.	Normal	No Syph	Improved	,,
M.	47	2nd part Innom--inate Artery	A.B. in 7th space.	Visible pulsating tumour of man--ubrium and inner half of right Clavicle, voice husky, tumour has dull note & systolic bruit, Dyspnoea Veins of neck distended, Episternal notch pulsation, & double bruit heard	Normal	No Syph.	Improved	,,
M.	47	1st & 2nd	Normal.	Tracheal Stridor, Dyspnoea on exer--tion, increased area of dulness over Aorta.	Normal	No Syph	Improved	Dr Bruce

BROMPTON HOSPITAL FOR CONSUMPTION.

----------o0o----------

CLINICAL RECORDS 1890 - 1896 (inclusive).

----------o0o----------

BROMPTON HOSPITAL FOR CONSUMPTION.

----------oOo----------

CLINICAL RECORDS 1890 - 1896 (inclusive).

----------oOo----------

SEX	AGE	PART OF AORTA	STATE OF HEART.
M.	42	1st & 2nd	Normal, no bruit.
M.	47	2nd & Innomin--ate Artery.	A.B. 6th space X.N.L. Diffuse im--pulse, Systolic Bruit & Accentua--ted Second.
M.	42	2nd & 3rd	Enlarged 21 oz. Double Aortic Bru
M.	57	2nd part	Some Hypertrophy, Systolic Bruit at Apex, Accentuated Second,
M.	50	1st & 2nd & 3rd	Atrophied, no Bruits, Accentuated Second
M.	33	1st part	Normal Accentuated Second.
M.	60	2nd part	Normal Accentuated Second.

BROMPTON HOSPITAL FOR CONSUMPTION.

----------oOo----------

CLINICAL RECORDS 1890 - 1896 (inclusive).

----------oOo----------

EFFECTS OF THE ANEURYSM	LARYNX	CAUSE	RESULT	OBSERVER
Swelling in the 1st & 2nd right inter -space & over right border of Sternum. Dull note, Systolic Bruit, Faint Dyas -tolic, Stridulous Respiration, husky voice, & Cough.	No note	Syphilis	I.S.Q.	Dr Bruce
Visible Tumour over inner end of left Clavicle, of Manubrium & both left & right interspaces; Veins distended Dulness all over extending to right & left; Systolic Bruit, Tracheal tug--ging, thrill felt over tumour.	No note	Syphilis	Improved	,,
Pressure on Trachea, no notes extant vide the P.M. records	Normal	No Syph	Post Mortem G. 107	,,
Pressure on the left Bronchus & left Subclavian Husky voice, Dysponea, L. Pupil > R.Pupil	Normal	No Syph.	Improved	,,
Dulness over the Manubrium down to base of heart & extending to right & left, Brassy Cough, Haemoptysis, Dyspnoea, Husky voice, Tracheal Tug--ging.	No note	Syphilis & Alcohol	Post Mortem H.104	,,
Dulness over 1st 2nd 3rd Interspaces right of Sternum, Pulsation felt Double Bruit heard.,	No note	Syphilis	Post Mortem G.299	,,
Pulsation over the Manubrium, 1st interspace on both sides, Dull note here, Double Bruit, R.Pupil > L.Pupil Veins on Chest dilated.	L.V. Cord in Cadaver--ic Po--sition	? Syph--ilis	Improved	,,

BROMPTON HOSPITAL FOR CONSUMPTION.

----------oOo----------

CLINICAL RECORDS 1890 - 1896 (inclusive).

----------oOo----------

SEX	AGE	PART OF AORTA	STATE OF HEART.	EFFECT OF THE ANEURYSM	LARYNX	CAUSE	RESULT	OBSERVER
M.	39	1st & 2nd	A.B. in 5th space I.N.L. Systolic at the Apex, Accentuated Second.	Pulsation of the right Clavicle Sternal end & in Episternal notch & Manubrium, R.Pupil > left Pupil thrill felt over the Tumour, Dull note all over this area & a double bruit.	No note	Syphilis	Improved	Dr Bruce
M.	40	2nd part	Normal in size, Double Bruit at Apex & base.	Dysphagia, Brassy Cough, Severe Stridor Double Bruit over Manubrium, R.Pupil > L.Pupil, Dulness & some pulsation down left side of Sternum, also of the left Clavicle, Husky voice	LVC Impaired Movement	None	Relieved	,,
M.	51	1st part	Normal in size, Systolic at Apex Reduplicatdd First, Accentuated Se	Pulsation felt in 1 2 & 3 right inter spaces, Dull note & a Systolic Bruit audible.	L.V.C. Impaired Movement	Syphilis	Improved	,,
M.	43	1st & 2nd part .	A.B. in 5th space I.N.L Accentuated Second	Pressure on right Bronchus, Veins of neck engorged, visible pulsating Tumour to the right of & over the Manubrium, also over the 1st 2nd & 3rd right ribs. No Bruit Heard Very dull, Tracheal tugging, Husky voice.	No note	No Syph.	Releived	,,
M.	60	2nd & Innominate Artery	Slight Hypertrophy, Systolic Bruit at Apex, Accentuated Second.	Much Dyspnoea Pulsation in veins of neck; No further Notes; Case unfinished.	No note	Syphilis	Improved	Dr Acland
M.	26	1st 2nd & 3rd	Hyperyrophied, Systolic Bruit at Apex.	Systolic murmur & thrill all over chest, Angina pains in left shoulder & left arm, no pressure symptoms except the neuralgic pains	No note	None	Improved	Dr William
M.	57	1st & 2nd parts	Hypertrophied, Double Bruit at Apex.	Dulness over the Manubrium & on either side of it, Double Bruit over the dull area, Right Pulse greater than the left Pulse.	No note	? Syphilis	Post Mortem H.163	,,

BROMPTON HOSPITAL FOR CONSUMPTION.

----------oOo----------

CLINICAL RECORDS 1890 - 1896 (inclusive).

----------oOo----------

BROMPTON HOSPITAL FOR CONSUMPTION.

----------oOo----------

CLINICAL RECORDS 1890 - 1896 (inclusive).

----------oOo----------

SEX	AGE	PART OF AORTA	STATE OF HEART.	EFFECTS OF THE ANEURYSM	LARYNX	CAUSE	RESULT	OBSERVER
M.	42	2nd part	A.B.X.N.L.very diffuse, Accentuated Second, ringing first	Tenderness & impaired note in 1st & 2nd right spaces, Systolic Bruit here Cough, Haemoptysis, Dyspnoea	No note	No Syph	Improved	Dr William
M.	43	2nd & 3rd parts	A.B.in 5th space I.N.L., Systolic Bruit at Apex, Accentuated Second.	Bulging of Manubrium & 1st & 2nd right & left Costal Cartilages & spaces, Pulsation felt, Clavicles also bulges & pulsate, also bulging & a pulsation in Episternal notch. Dull note; marked Tracheal tugging, husky voice, Brassy Cough, R.Pulse greater than L.Pulse, Systolic Bruit over Tumour.	L.V. Cord in Cadaveric position	Alcohol strain	Improved	,,
M.	51	1st part	A.B.in 6th space I.N.L., Double Bruit at the Apex	Bulging of the 2nd & 3rd right spaces & Cartilages, Visible Pulsation marked thrill, very tender, Double Bruit, R.Bupil > L.Pupil	No note	Alcohol Syphilis	Improved	,,
F.	53	3rd part	Slightly enlarged, Systolic Bruit at Apex, Accentuated Second.	Pain in the back under left Scapula Dyspnoea, R.Pupil > L.Pupil, Constant pain in back	No note	None	Improved	Dr Biss
F.	44	1st & 2nd parts	Hypertrophied, Double Bruit at Apex & at base.	Much Dyspnoea; no further notes	No note	? Syph.	Post Mortem (K/64)	,,
F	36	1st part	A.B.in 5th space, I.N.L. Systolic Bruit at Apex, Accentuated Second	Bulging of Manubrium Dilated Venules over it Dull note Systolic Bruit	No note	None	Improved	Dr Acland
M.	42	3rd part	Overlapped by lungs, Obscured sounds, A.B. not felt	Pressure on L.Bronchus, Aphonia Cough Constant Stridor, Pulsating Tumour 1st R.space & Manubrium, Dull note here, R.Pulse > L.Pulse, R.Pupil > L.Pupil	L.V.cord paralysed	None	P.M. H.136	Dr Fowler

BROMPTON HOSPITAL FOR CONSUMPTION.

----------oOo----------

CLINICAL RECORDS 1890 - 1896 (inclusive).

----------oOo----------

BROMPTON HOSPITAL FOR CONSUMPTION.

----------oOo----------

CLINICAL RECORDS 1890 - 1896 (inclusive).

----------oOo----------

BROMPTON HOSPITAL FOR CONSUMPTION.

----------oOo----------

CLINICAL RECORDS 1890 - 1896 (inclusive).

----------oOoOo----------

SEX	AGE	PART OF AORTA	STATE OF HEART.	EFFECTS OF THE ANEURYSM	LARYNX	CAUSE	RESULT	OBSERVER
M.	51	3rd part	A.B.in 5th space Systolic Bruit at Apex, Accentuated Sec.	Pressure on left Bronchus, Dyspnoea on exertion	No note	None	Improved	Dr Fowler
M.	47	1st part	A.B.in 5th space I.N.L. Systolic bruit.	Prominent pulsating Tumour, Sec. Right Interspace, dull note, Systolic Bruit	No note	Strain	Improved	,,
F.	55	2nd & 3rd parts	A.B.in 5th space I.N.L. Double Bruit at base.	Bulging of Sternum, partial obstruc--tion left com. Carotid, impaired note left of Sternum, Systolic Bruit	No note	None	Relieved	,,
F.	29	1st part pulmonary Art.	Hypertrophied left Venticle, impulse in 4th & 5th space, Systolic bruit at Apex, very Accentuated Sec.	Cyanosis of face very severe, excess--sive Dyspnoea, marked clubbing of fingers	No note	No note	P.M.	,,
F.	30	2nd part	Heaving Precordium Systolic at Apex, Double bruit at base.	Bulging of Sternum & to the right dilated venules over it, pulsation felt & very tender, dull note Systol--ic Bruit, Dyspnoea bad, Tumour the size of an orange	No note	None	P.M. I. 228	,,
M.	37	2nd & 3rd parts	Displaced to the left, faint Sys--tolic at Apex.	Husky voice, Compression of Trachea & left Bronchus, prominence over man--ubrium & to the left with heaving pulsation, tumour is decidely to left side of chest, dull note over it, Trachea--al tugging , Dysphagia.	L.V.C. in Cadaveric position.	Syph.	Improved	,,
M.	28	2nd & 3rd parts	No note	Pulsation inner end L.Clavicle & in first left interspace, dull note Brassy cough, Tracheal tugging, veins dilated over tumour, loud stridulops breathing.	L.V.C. in Cadaveric position	Syph. & Alcohol	P.M. K.76	,,

BROMPTON HOSPITAL FOR CONSUMPTION.

----------oOo----------

CLINICAL RECORDS 1890 - 1896 (inclusive).

----------oOo----------

SEX	AGE	PART OF AORTA	STATE OF HEART.
M.	54	2nd part	Area of Dullness obscured, no Bruits, Epigastric pulsation.
M.	46	2nd & 3rd parts	Displaced to the right, no bruits Accentuated Sec.
M.	43	2nd & 3rd parts	A.B.in 5th space I.N.L. Systolic bruit at Apex, Accentuated Sec.
F.	33	1st & 2nd parts	Normal in size, A.B.obscured, second sound not accentuated.
F.	53	2nd part	A.B.in 5th space I.N.L. Double bruit at Apex & in Aortic area.
M.	54	2nd part	A.B.in 6th space X.N.L. Systolic bruit at Apex.
M.	45	1st & 2nd part.	A.B.in 6th space X.N.L. Double bruit at Apex

BROMPTON HOSPITAL FOR CONSUMPTION.

----------oOo----------

CLINICAL RECORDS 1890 - 1896 (inclusive).

----------oOo----------

EFFECTS OF THE ANEURYSM	LARYNX	CAUSE	RESULT	OBSERVER
Tracheal Stenosis, Dyspnoea & Stridor marked. R.lung dull back & front	R.V.C. in Cadaveric position.	Syphilis	I.S.Q.	Dr Fowler
R.lung dull all over Systolic Bruit in 2nd right space Diffused pulsation here Dyastolic in 1st space(L) much Dyspnoea.	L.V.C. impaired movement	? Syph.	Improved	Dr Roberts
Dulness over upper half of Sternum & to the right & left sides, veins of chest dilated, Systolic Bruit, com-pression of Trachea & of Subclavian Artery, Bruit Stridulous.	No note	Syphilis	P.M. F.125	,,
Cyanosis, heaving pulsation in 2nd & 3rd right spaces & of the Manubrium visible, Dull note Systolic Bruit Tracheal tugging, L.Pulse > R.pulse.	No note.	Strain	Improved	,,
Prominent pulsating tumour in upper half of Sternum, dull note Systolic bruit.	No note.	Hard work. in a laundry.	Improved	Dr Kidd.
Pressure on Trachea Brassy cough Dysp-noea, & some Dysphagia, visible pulsa-tion in upper part of Sternum & to the left, Systolic Bruit & dull note over it.	No note.	Syphilis.	Improved	,,
Visible pulsation in left space dull-ness on right side of Manubrium going across it to the left side, Systolic Bruit, Tracheal tugging, L.Pupil > R. Pupil.	L.V.C. para-lysed	Syphilis	Improved	,,

BROMPTON HOSPITAL FOR CONSUMPTION.

----------oOo----------

CLINICAL RECORDS 1890 - 1896 (inclusive).

----------oOo----------

SEX	AGE	PART OF AORTA	STATE OF HEART.
M.	38	1st & 2nd parts	A.B., X.N.L. Accentuated Sec. a weak first, no bruits.
F.	66	2nd & 3rd	A.B.in 5th space X.N.L. Systolic bruit & a booming Sec. in Aortic area.
M.	52	2nd part	Area of dulness obscured by Emphy--sema of both lungs.
M.	52	1st & 2nd	Obscured A.B.not felt, faint sound & no Bruits.
M.	63	1st part	A.B.in 5th space I.N.L. faint Sys--tolic bruit at Apex, 2nd sound Accentuated.
M.	37	1st & 2nd parts	A.B.in 6th space I.N.L. double bruit at Apex.

BROMPTON HOSPITAL FOR CONSUMPTION.

----------oOo----------

CLINICAL RECORDS 1890 - 1896 (inclusive).

----------oOo----------

EFFECTS OF THE ANEURYSM	LARYNX	CAUSE	RESULT	OBSERVER.
Tracheal Stenosis Brassy cough,Stri--dor, some Dysphagia,visible tumour to right of Manubrium pulsation, Dyastolic shock,dull note all over tumour extending along right clavicle tracheal tugging present.	Normal	? Syph.	Relieved	Dr Kidd
Visible tumour in 1st 2nd & 3rd left spaces pulsating,Dyastolic shock Tracheal tugging very marked,Manu--brium pulsates,Episternal notch pulsates,dull note all over here, much Dyspnoea, R.Pulse > L.Pulse.	L.V.C. impaired movement.	Syph.	Improved	,,
Altered voice,Cough,marked Dyspnoea, marked Tracheal tugging, R.Pulse < L.Pulse, R.Pupil < L.Pupil.	Normal.	No Cause	I.S.Q.	,,
Dull area from right clavicle down to 3rd rib,Bronchial breathing & a Double Bruit here.	Normal	Syph.	I.S.Q.	,,
Visible tumour over 3 4 & 5 left ribs & over Sternum, it pulsates dull note,L.Pupil < R.Pupil.	Normal	Alcohol & hard labour.	Improved	,,
Bulging pulsating tumour on right side down to 4th rib, dull note Dyas--tolic thrill & Dyastolic Bruit.	Normal	Syph.	Improved	,,

BROMPTON HOSPITAL FOR CONSUMPTION.

----------oOo----------

CLINICAL RECORDS 1890 - 1896 (inclusive).

----------oOo----------

SEX	AGE	PART OF AORTA	STATE OF HEART	EFFECTS OF THE ANEURYSM	LARYNX	CAUSE	RESULT	OBSERVER.
M.	31	2nd part	A.B.not felt, sounds clear at Apex Systolic & Accentuated Sec. at base	Brassy cough, Tracheal tugging, pressure on left Bronchus, pulsation in the 2nd right space, Stridulous breathing, Systolic Bruit here & in 2nd space.	No note	Syph.	P.M. H.150	Dr Tathem
M.	46	1st & 2nd part	A.B.in 5th space, Systolic bruit at base, Accentuated Sec.	Dull note over Manubrium & 2nd & 3rd right interspaces, no visible pulsation, husky voice, brassy cough, Dyspnoea on exertion, Stridulous breathing sometimes.	L.V.C. paralysed	None.	P.M.	Dr Bruce
M.	61	1st & 2nd part	A.B.in 6th space I.N.L. no Bruits at Apex or base, Accentuated Sec.	Impaired note to right of Sternum & of Sternum, no bruit audible, wheazy cough, Stridulous respiration, R.Pupil > L.Pupil, attacks of Dyspnoea & Haemoptysis.	Normal	None	P.M. M.14	,,
M.	40	2 inches above the diaphragm.	Tilted forwards & to the right Systolic bruit at Apex & at base.	Constant gnawing pain in the back & left groin, dull area beneath the inferior angle of left Scapula & of left base of lung, weak breath sounds here.	Normal	None	P.M. M.17	,,

BROMPTON HOSPITAL FOR CONSUMPTION.

----------oOo----------

CLINICAL RECORDS 1890 - 1896 (inclusive).

----------oOo----------

ROYAL HOSPITAL FOR DISEASES OF THE CHEST.

----------oOo----------

CLINICAL RECORDS 1890 - 1896 (inclusive).

----------oOo----------

SEX	AGE	PART OF AORTA	STATE OF HEART.
M.	34	1st part	A.B.in the 5th space I.N.L. sounds obscured by wheazy respiration, Accentuated Sec. at the base.
M.	54	1st 2nd & 3rd parts	A.B.in 6th space X.N.L. Systolic bruit at the Apex, double bruit at base, Accentuated Second.
M.	56	2nd & 3rd parts	A.B.in 5th space I.N.L. no bruits heard, 2nd sound Accentuated.
M.	47	2nd & 3rd parts	A.B.in the 5th space X.N.L. double bruit at Apex, double bruit at the base , much Hypertrophy.
M.	51	3rd part	A.B.X.N.L . no bruit at Apex, Sys--tolic & pulmonary area,Second Accentuated.
M.	43	3rd part	Slightly enlarged, bruit at Apex faint double bruit at base.
M.	62	1st & 2nd parts	A.B.in the 6th space, X.N.L.Systo--lic at Apex, Accentuated Sec.
M.	43	1st part	Area of dulness obscured, A.B. not felt, double bruit at base.

ROYAL HOSPITAL FOR DISEASES OF THE CHEST:-

----------oOo----------

CLINICAL RECORDS 1890 - 1896 (inclusive).

----------oOo----------

EFFECTS OF THE ANEURYSM	LARYNX	CAUSE	RESULT	OBSERVER.
Brassy cough, Dyspnoea on exertion, Dysphagia & Haemoptysis, dull note along right border of Manubrium & upper 3 right ribs.	Normal	No Syph.	Improved	Dr Finlay.
Pulsation in the vessels of the neck impaired note over the Manubrium & on either side of it, pain in the back.	No note	None	Death.	Dr Gilbart Smith.
Visible heaving pulsation over Manu--brium & to the left side, superfici--al veins dilated, impaired note over this area, brassy cough, stridulous respiration.	Normal	Syph.	Death	Dr Hensley
Visible pulsation in 1st left space no thrill, dull note over 2nd left cartilage, Systolic bruit, some Dys--phagia.	L.V.C. para--lysed.	Rheumatic fever.	Improved	,,
Visible pulsation of a tumour over 1st 2nd & 3rd left ribs, dull note over it, Bronchial breathing, Sys--tolic bruit,	Normal	None	Improved	,,
Impaired note to left of Sternum, loud double bruit over it.	No note	Rheumatic fever.	Improved	,,
Dull note over Manubrium & down the adjoining right spaces for $3\frac{1}{4}$ inches wide, marked thrill & pulsation here, double bruit over it.	Normal	Alcohol	Improved	Dr White
Clinical features obscured by severe bronchitis & Emphysema.	No note	?--	Improved	Dr Gilbart Smith.

SEX	AGE	PART OF AORTA	STATE OF HEART.	EFFECTS OF THE ANEURYSM	LARYNX	CAUSE	RESULT	OBSERVER.
F.	49	1st part	A.B.in 5th space X.N.L. double bruit at base, Sec sound Accen--tuated.	Visible pulsation in Second right space, thrill felt, dull note over it & a double bruit heard.	Normal	Hard work.	Improved	Dr White.
M.	37	2nd part	A.B.not felt, area of dulness normal, no bruit audible, Accen--tuated Sec.	Pressure on Trachea, brassy cough, husky voice, impaired note over Man--ubrium.	Normal	Syphilis	Improved	Dr Calvert
M.	64	1st & 2nd part	A.B.X.N.L. no bruit heard, Accentuated Sec.	Hoarse cough, altered voice, pres--sure on right Bronchus, visible pul-sation in 2nd & 3rd right spaces, dull note, Systolic bruit audible.	L.V.C. impaired movement.	No note	Ruptured into R. Bronchus.	Dr Davies.
M.	57	Innominate artery	A.B.in 5th space I.N.L. no bruit here, dyastolic at base.	Visible pulsation of right clavicle & below it, dull note, loud Systolic bruit, some dulness over Manubrium, R.Pupil > L.Pupil, husky voice.	Normal	None	Improved	Dr Hensley
M.	51	2nd part	A.B.not felt, area of dulness normal, Accentuated Sec.	Tracheal Stridor, metallic cough, Dyspnoea on exertion, impaired note over 1st & 2nd left ribs & spaces, right pulse > left pulse.	Bilateral abductor paralysis.	None.	Improved	,,
M.	53	1st part	A.B.in 5th space I.N.L. faint Systolic at Apex, Accentuated Sec.	Dull note over right half of Manu--brium & 1 & 2 right ribs & spaces visible pulsation & systolic bruit here.	Normal	Alcohol	Improved	,,
F.	58	Innominate artery	A.B. in 5th space X.N.L. dyastolic bruit at Apex also heard at base, very accentuated pulmonary Sec.	Visible pulsating tumour above right clavicle & of clavicle, veins in upper part of chest dilated, dull note over tumour.	No note	None	Improved	,,
M.	58	1st part	A.B. in the 5th space I.N.L. Accen--tuated Sec.	Impaired note over Manubrium & to right side of it, visible pulsating tumour here, systolic bruit, brassy cough.	Normal	Syphilis	Improved	Dr Oswald Browne.
M.	42	2nd part	A.B. in the 7th space I.N.L no bruits audible here.	Manubrium is very prominent, super--ficial venuels dilated, dull note elicited, R.Pupil > L.Pupil.	L.V.C. para--lysed.	Syphilis	Death	Dr White
M.	39	2nd & 3rd parts	A.B. not felt, area of dulness normal , systolic at apex & base. Accentuated sec.	Dull area extending from base of heart to inner half left clavicle & involving Manubrium, systolic pulsa--tion seen & felt here in 2nd left space. R.Pulse > L.Pulse.Husky voice.	L.V.C. para--lysed.	Syph. & Alcohol	Improved	,,

SEX	AGE	PART OF AORTA	STATE OF HEART.	EFFECTS OF THE ANEURYSM	LARYNX	CAUSE	RESULT	OBSERVER.
M.	54	1st & 2nd parts	Area of dulness normal, sounds are distant & faint, Second is Accen--tuated at the base.	Impaired note over Manubrium & to the right side in upper 3 spaces, well marked pulsation, no bruit, heart sounds very distinct, altered voice, brassy cough, Tracheal tug--ging well marked, Dyspnoea.	L.V. cord para--lysed	No note	I.S.Q.	Dr Gilbart Smith.
M.	41	1st & 2nd parts	A.B. in 5th space I.N.L. no bruits at the Apex, systolic at the base & Accentuated Sec.	Pulsation of the Manubrium & of right clavicle visible, dull note in this area & upper 3 right spaces, rough systolic heard here, Accentuat--ed Sec. very pronounced.	Normal	Syph.	Improved	Dr Calvert
M.	50	Lst & 3rd & descending thoracic.	A.B. not felt, sounds not audible Heart displaced to right side of chest.	Visible swelling on the right side over 2nd 3rd & 4th ribs large as a big orange, left chest bulging a good deal, no pulsation seen or felt, dull note over tumour & all over left side, no bruits.Heart sounds very distinct.	L.V.C. para--lysed	No cause.	P.M. 1080	Dr Hensley
M.	53	1st & 2nd Innominate Artery.	Hypertrophied, systolic at base & Accentuated Sec.	Brassy cough Stridor marked, voice altered, heaving pulsation over Men--ubrium & to the right side & of right clavicle, dull note here, pul--sation in episternal notch, systolic bruit.	L.V.C. impaired movement	None.	P.M. 1012	Dr Gilbart Smith.

BROMPTON HOSPITAL FOR CONSUMPTION.

----------oOo----------

(POST-MORTEM RECORDS 1890 - 1896).

----------oOo----------

NO. IN THE REGISTER	SEX	AGE	PART OF AORTA	STATE OF HEART.
F.125	M	43	2nd & 3rd	Very small.
F.143	M.	42	1st part	Left ventricle slightly hypertrophied.
F.165	M.	48	1st & 2nd	No hypertrophy.
G.107	M.	42	3rd part	Much enlarged, 21 ounces.
G.256	M	45	1st & 2nd part.	Somewhat enlarged.
G.299	M.	33	1st part	No note.
H.104	M.	50	1st 2nd & 3rd.	Very small.

BROMPTON HOSPITAL FOR CONSUMPTION.

----------oOo----------

(POST-MORTEM RECORDS 1890 - 1896).

----------oOo----------

DESCRIPTION.

A sacular aneurysm size of a tangerine on posterior wall pressing on trach -ea & Left Subclavian artery, obliterating the latter. Origin of innom- -inate artery involved. Aneurysm runs up right side of trachea, causing ulceration; manubrium & Sternal end of clavicle a little eroded.

Sacular aneurysm size of a large orange, immediately under sternum & two first right spaces. Trachea, much ulcerated, and perforated at 9th & 10th rims, the cause of death. Right bronchus congested; it is on the dextro-anterior aspect of aorta, & communicated with same by a whole the size of a penny.

General dilatation of the ascending & transverse portion, with sacular aneurysm at the commencement of the descending part; it grows from the left wall, & is eroding the left bronchus; it occupies the upper half of upper lobe of left lung; pressure on 4, 5, 6, & 7th dorsal vertebrae, causing necrosis. Recurrent laryngeal involved.

Junction of 3rd part with descending thoracic aorta a sacular aneurysm size of a tangerine; it grew from the posterior wall, and pressed upon & bulged into the anterior wall of trachea, but did not rupture.

Fusiform aneurysm from immediately above the valves to the origin of left subclavium. Left common carotid is involved, being obliterated.

Globular swelling growing to the left size of a man's fist, & going behind pulmonary artery; communicates with aorta just above the posterior Sinus Valsava by an opening size of a florin; it surrounds & incorporates left bronchus & left pulmonary vein.

Fusiform of entire arch. On the dextro-anterior aspect of ascending limb is a sacular aneurysm size of a walnut, a second one is found arising from posterior wall of transverse part, and involves left bronchus, into which it ruptured.

NO. IN THE REGISTER	SEX	AGE	PART OF AORTA	STATE OF HEART.	DESCRIPTION.
H.136	M	42	1st 2nd & 3rd	Slightly enlarged.	First & second parts much dilated, a very large sacular aneurysm of de-scending limb; it pressed on the 4th 5th & 6th vertebrae & intervertebral disks, these forming its posterior wall; in front it pressed on the left bronchus, left recurrent laryngeal nerve also involved; L.V.cord paraly-sed.
H.150	M	31	2nd part	No note.	Entire arch dilated; sacular aneurysm on posterior wall of transverse part; point of communication with aorta was its widest, being very shal-low; it pressed upon & ruptured into left bronchus.
H.163	M	53	1st part	Dilated; left ventricle is hypertrophied.	General dilatation of arch & sacular dilatation of 1st part which pressed upon right auricle, & communicated with it by a small opening.
H.208	M	37	1st part	Enlarged a little, left ventricle a little hypertrophied.	First part much dilatated, a very large sacular aneurysm grew from its left wall just above the left coronary aretry; its aperture of communi-cation with aorta was 7 inches in circumference; it passed behind pul-monary artery, & caused much pressure on the left auricle.
H.238	M	50	1st part	Much dilated & hyper-trophied.	Entire aorta is much dilated, but first part is excessively so, & most marked on the right wall, becoming sacular here.
I.114	M	53	2nd & 3rd	No note.	Sacular aneurysm passing backwards & eroding two of the dorsal vertebrae commences at distal side of origin of innominate artery, & ended at root of left lung; much pressure on left bronchus.
I.139	M	49	1st part	Large & hypertrophied.	Excessive dilatation of ascending limb, no further notes.
I.228	F	30	2nd part	Left ventricle Hypertro-phied.	Sacular aneurysm of right wall of arch, practically an aneurysm of the inn-ominate artery; it grew forward, pressed upon the Manubrium & eroded it
I.233	M	54	1st & 2nd part	Left ventricle dilated & a little hypertrophied	General diffuse dilatation of the ascending & transverse parts; it pre-forated into the pulmonary artery, about 1 inch above the pulmonary valve, aperture admitted tip of little finger.
K.22	F	29	1st part of pulmonary artery.	Patent foramen Ovale. Hypertrophied & dilata-tion of right heart.	Aneurysm was intra & extra pericardial in position; both the main branches of pulmonary artery were greatly thrombosed; aneurysm was fusi-form & full of laminated clot; artery was very atheromatous throughout its entire course through the lungs; aneurysm commenced immediately above the valves & ended at the bifurcation of the artery.
K.36	M	43	1st part	Hypertrophied & dilated.	Fusiform dilatation of ascending limb most marked on its dextro anterior surface.
G ?.	M	46	1st & 2nd	No note.	General dilatation of the ascending & transverse parts, the latter por-tion causing much pressure upon the trachea.

NO. IN REGISTER	SEX	AGE	PART OF AORTA	STATE OF HEART.	DESCRIPTION.
K.76	M	28	1st part	Weight 1o oz. Valves healthy, L.Venticle a little hypertrophied.	Saculated aneurysm size of a large orange on posterior wall, 1½ inches above valve; it pressed upontthe bifurcation of trachea, & both bronchi, ruptured into right bronchus, S.V.Cava involved; broncho pneumonia pre-sent from pressure on the lung.
K.161	F	44	1st & 2nd	Weight 21 oz. left ven-tricle hypertrophied	Sacular aneurysm size of an orange on posterior wall just above the valves compression of trachea & of oesophagus, transverse part is also dilated.
L. ?.	M	36	Innominate artery.	Weight 10½ oz. right side dilated, right auricle specially dila-ted, pulmonary artery full of clot.	Globular aneurysm size of a foetal head & springing from anterior wall of innominate artery, & growing up in front of its bifurcation; left innominate vein is occluded; S.V.Cava compressed; trachea is pressed upon just above bifurcation; commencing aneurysm on dextro posterior wall of ascending limb.
L. ?.	M	51	1st part	Valves atheromatous, right heart a little dilated.	Sacular aneurysm the size of a tangerine on the left posterior wall, 1½ inches above the valves; it compressed the left auricle, pulmonary veins & S.V.Cava.
M.14	M	61	1st & 2nd	Left ventricle hyper-trophied, aortic valves diseased, but competent.	Fusiform dilatation of ascending & transverse part, small sacular aneu-rysm at junction of ascending & transverse part, on posterior wall pres-sing on right bronchus. Another sacular aneurysm, the size of a walnut on posterior wall of transverse part is pressing upon & ulcerating the right wall of the trachea ½ an inch above its bifurcation.
M.17	M	40	2 inches above diaphragm	Weight 11 oz. Valves healthy.	Large sacular aneurysm on posterior wall 2 inches above opening in diaph-ragm, pressure upon 8th, 9th, 10th, 11th dorsal vertebrae, causing ero-sion and necrosis; perforation into left pleural sack.

ROYAL HOSPITAL FOR DISEASES OF THE CHEST :-

----------oOo----------

(POST-MORTEM RECORDS 1892 - 1896).

----------oOo----------

NO. IN REGISTER	SEX	AGE	PART OF AORTA	STATE OF HEART.
25	M	48	1st & 2nd parts	Hypertrophied left ventricle.
28	M	43	1st part	Weight 26¼ oz.Hypertro--phied & dilated, L.Ven--tricle very Hypertrod.
43	M	43	1st part	Wt. 1 lb. 13¼ oz. great hypertrophy.
84	M	37	2nd part	Wt. 12¼ oz. some hyper--trophy of left ventricle
105	M	57	1st part	Normal.
206	M	42	2nd part	Left ventricle slightly hypertrophied.
?.	M	50	1st 2nd & 3rd	Particularly small
?.	M	53	1st 2nd & innominate	Slight hypertrophy of of left ventricle.

ROYAL HOSPITAL FOR DISEASES OF THE CHEST :-

----------oOo----------

(POST-MORTEM RECORDS 1892 - 1896).

----------oOo----------

DESCRIPTION.

Aorta is very atheromatous, ascending & transverse portions dilated, sac--ular aneurysm the size of a large orange, junction of the latter with the descending limb, & causing pressure on upper lobe of left lung.

Ascending limb greatly dilated, sacular aneursym on its posterior wall, going backwards & inwards, pressure on pulmonary artery nearly occluding it.

Aorta very atheromatous & ulcerated, the dilatation of right wall extends for 1½ inches above the valves, being nearly sacular.

Sacular aneurysm on posterior wall size of a tangerine orange; it is growing backwards & inwards, pressing on trachea, aorta very atheromatous.

Sacular aneurysm size of a large orange on anterior wall of ascending limb commencing ½ an inch above the valves ; anterior wall of sac is just under Manubrium sterni.

Two aneurysms present; sacular one size of a tangerine, involving innom--inate artery; the second is at commencement of descending limb, sacular & large; it opened by two openings into aorta, the first pressed on the trachea, & the latter pressed heavily on the left bronchus.

Two aneurysms, the first globular, and as large as a foetal head grew from ascending limb, & was under 2nd, 3rd, & 4th right ribs, and Manubrium causing much necrosis; the second commenced at distal side of innominate artery, & was one huge blood sack down to 1 inch above the opening of the diaphragm; it occupied entire left chest, the left lung being collapsed

Fusiform dilatation of entire arch, descending limb not involved; sacular aneurysm of innominate artery; it was of great size, & pressed on trachea where it perforated & ruptured; the right sterno clavicular articulation was much eroded.

ANALYSIS OF THE SYMPTOMS.

----------oOo----------

	Vocal Cord Paralysis.	Dysphagia.	Stridor.
Frequency of Occurrence	46•2%	7•7%	17•6%
1st part of Aorta	1	1	1
1st & 2nd parts of Aorta	5	1	4
1st 2nd & 3rd parts	1	-	1
2nd part of Aorta	7	3	4
2nd & 3rd parts of Aorta	8	2	5
3rd part of Aorta	1	-	1
1st & 3rd parts of Aorta	1	-	-
Innominate Artery	-	-	-

----------oOo----------

ANALYSIS OF THE SYMPTOMS.

----------oOo----------

Brassy Cough.	Inequality of Pulses.	Inequality of Pupils	Tracheal Tugging	Altered Voice.
26•4%	16•5%	16•5%	14•3%	18•7%
3	2	2	-	1
7	3	3	5	5
2	-	-	1	1
7	3	6	3	6
4	6	1	4	3
1	1	2	-	-
-	-	-	-	-
-	-	1	-	1

----------oOo----------